

A mathematical Model of SIR Transmission Dynamics with Homotopy Analysis Approach

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Abstract

In this paper SIR(Susceptible-Infected-Recovered) epidemic model is formulated and analysed. Existence and stability of the Disease Free Equilibrium (DFE) of the model is discussed in detail. The basic reproduction number R_0 of the model is computed and it is established that the disease free equilibrium of the model is globally asymptotically stable for $R_0 < 1$. Homotopy Analysis Method (HAM) is used to solve the model. Semi-analytical results obtained by HAM have been compared with the numerical solution and are found to be in good agreement. Finally, various simulations are done to discuss the solution.

Mathematics subject classification. 92B05; 92D25; 92D30; 93D05; 34K20; 34K25.

Keywords: Epidemic, Homotopy Analysis Method, Equilibrium, Basic reproduction ratio.

1 Introduction

Closed form solutions of non-linear differential equations are sometimes not easy to get. In recent time, there exist various software such as MATLAB, MAPLE, MATHEMATICA, MAXIMA, OCTAVE, SCILAB etc. The routines built into these software may be capable of gaining understanding of the dynamics of the problem. But the biology of infectious diseases in cases where it is governed by the nonlinear differential equations may not yield good results or may even fail completely due to various problems like singularities, stiffness or multiple solutions the numerical approach etc. HAM was first discovered in 1992 by Liao Shijun in his P.hd dissertation and further modified in 1997 to introduce a non-zero auxilliary parameter, referred to as the convergence-control parameter to construct a homotopy on a differential system in general form. In this paper, the existence and stability of the model is carried out and the Homotopy Analysis Method is applied to solve the SIR epidemic model. The HAM results obtained are compared with the numerical results and found in strong agreement with the numerical results. At first, a mathematical model is formulated for SIR transmission dynamics and then apply the HAM to find the semi-analytical solution. This work presents a semi analytical technique viz. the Homotopy Analysis Method (HAM) which has been applied to study the solution of the epidemic model. This method employs the concept of the homotopy from topology to generate a convergent series solution for nonlinear systems which is enabled by utilizing a homotopy-McLaurin series to deal with the nonlinearity in the system. The strength of the HAM to naturally exhibit convergence of the series solution is strange in most analytic and semi-analytic approaches to nonlinear PDEs[13]. In the recent time, Khan et.al[12] used the HAM approach to solve the SIS and SIR models of Kermack and Mckendrick[7]. Motsa et.al[10] extended the work of Khan et.al[8] to solve the SIR epidemic model in the presence of constant vaccination strategy. Motsa[11] also applied the HAM to solve the SIRS epidemic model and obtained an explicit analytic solution of the coupled nonlinear differential equations describing the epidemic model obtained and comparison were made with the numerical results which shows that the two results are in good agreement. M. Sajid et.al [12] studied a new approach for solving SIR epidemic model using HAM. His new approach was based on dividing

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the entire domain into subintervals. The paper is organized as follows: in section 2, we present the model formulation. In section 3, we discussed the qualitative analysis of the model and the derivation of the reproduction number. In section 4, we present the Homotopy Analysis Approach to non-linear system. . While, in section 5, we present the solution of the malaria transmission model by HAM and In section 6, we present the Numerical results and discussion. In section 7, we discussed the conclusion and possible extensions and finally, the references are presented.

2 Model Formulation

A population comprising of three kinds of individuals whose numbers are denoted by S(Susceptible human), I(Infected human) and R(Recovered Human) is been considered. The Susceptible human ($S(t)$) is the number of susceptible human at time t i.e those who are vulnerable, who are yet to have the disease but have the tendency of having it, Infected human ($I(t)$) is the population of the infected and infectious, those who have the disease and can transmit it to others while the Recovered human ($R(t)$) is the population of the removed, those who cannot get the disease or transmit it either because they have got natural immunity, or they have recovered from the disease and immune from re-infection or they have been placed in isolation or they have died.

The population of susceptible humans is generated through reduction by the rate of transmission β such that the rate of change of population of susceptible human is given by:

$$\frac{dS}{dt} = -\beta SI, \quad \beta > 0 \quad (1)$$

The rate of change of the population of infected human is increased by the rate of transmission β and reduced by the rate at which the infected population become isolated γ . Hence it is given by:

$$\frac{dI}{dt} = \beta SI - \gamma I, \quad \beta > 0, \gamma > 0 \quad (2)$$

The population of recovered human is generated by the rate at which the infected population become isolated. Hence it is given by:

$$\frac{dR}{dt} = \gamma I \quad (3)$$

Hence the above formulation and assumptions together leads to the following System of ordinary differential equations:

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI \\ \frac{dI}{dt} &= \beta SI - \gamma I \\ \frac{dR}{dt} &= \gamma I. \end{aligned} \quad (4)$$

subject to the initial conditions

$$S(0) = S_0, I(0) = I_0, R(0) = R_0.$$

We described the associated model variables 1

Variables	Description
S	Susceptible Human
I	Infected Human
R	Recovered Human

Table 1: Table showing the Variables in the model.

(i) Susceptible: Those who do not have the disease(illness) but can catch it. They are the vulnerable group.

(ii) Infected : Those who currently have the disease(illness) and are contagious . They are infectious and infective.

(iii) Those who have recovered from the illness(disease) and are immuned. They could also stand for the removed class which means those who have removed from the disease either through death/immunity.

We assume that the population we are considering here is large but fixed in size and confined geographically well defined location e.g population of people in a classroom or boarding school. The population can be subdivided into three distinct compartments.

3 Qualitative Analysis

3.1 Positivity of the solution

Theorem 1.: Suppose the initial data $S \geq 0, I \geq 0, R \geq 0$, then the solutions $(S(t), I(t), R(t))$ of the SIR model (4) are non-negative for all $t > 0$. Therefore,

$$\limsup_{t \rightarrow \infty} N(t) \leq \text{Constant} \quad (5)$$

such that $N = S + I + R$.

Proof. We let $D = \sup\{t > 0 : S(t) > 0, I(t) > 0, R(t) > 0\}$.

Since the variables $S(0) > 0, I(0) > 0, R(0) > 0$ then, $D > 0$. If $D < \infty$, then S, I, R is equal to zero at D . It follows from the first equation of the system (4), that

$$\frac{dS}{dt} = -\beta SI$$

Therefore,

$$\frac{d}{dt} \left\{ S(t) \exp \int_0^t [(\beta I(\tau))] d\tau \right\} \geq 0.$$

We solve the inequality to obtain

$$S(t) \exp \int_0^t [(\beta I(\tau))] d\tau - S(0) \geq 0.$$

Therefore $S(t)$ yields

$$S(t) \geq S(0) \exp \int_0^t [(\beta I(\tau))] d\tau$$

Similarly

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Also

$$\frac{dI}{dt} + \gamma I - \beta SI = \frac{dI}{dt} + (\gamma - \beta S)I = 0$$

Therefore

$$\frac{d}{dt} \left\{ I(t) \exp \int_0^t [(\gamma - \beta S(\tau))] d\tau \right\} \geq 0.$$

We solve the inequality to obtain

$$I(t) \exp \int_0^t [(\gamma - \beta S(\tau))] d\tau - I(0) \geq 0.$$

Therefore $I(t)$ yields

$$I(t) \geq I(0) \exp \int_0^t [(\gamma - \beta S(\tau))] d\tau$$

Also,

$$\frac{dR}{dt} = \gamma I$$

Therefore,

$$\frac{d}{dt} \left\{ R(t) \exp \int_0^t [(0)] d\tau \right\} \geq \gamma I \exp \int_0^t (0) d\tau.$$

We solve the inequality to obtain

$$R(t) \exp \int_0^t [(\beta I(\tau))] d\tau - R(0) \geq \gamma I \exp \int_0^t (0) d\tau.$$

Therefore $R(t)$ yields

$$R(t) \geq R(0) \exp - \int_0^t [(0)]d\tau + \exp - \int_0^t (0)d\tau \times \gamma I \exp \int_0^t (0)d\tau > 0$$

3.2 Boundedness of the solution

Theorem 2.:Every solutions $(S(t), I(t), R(t))$ of the SIR model (4) is bounded. Therefore,from (5)

$$\limsup_{t \rightarrow \infty} N(t) \leq \text{constant}$$

such that $N = S + I + R$.

Proof. To proof boundedness, we note that $0 < R(t) \leq N(t)$ and $0 < S(t) \leq N(t)$. We add the model equation the SIR model (4) yield:

$$\frac{dN}{dt} = 0 \quad (6)$$

Then the

$$\limsup_{t \rightarrow \infty} N(t) \leq \text{Constant} \quad (7)$$

Therefore all solutions of model equation (4) are bounded. The feasible region for the total population is:

$D = \{S, I, R | S + I + R \leq \text{constant}, 0 \leq S \leq S(t), 0 \leq I \leq I(t), 0 \leq R \leq R(t)\}$ We define D as the positively invariant region with respect to the model equation(4) therefore the model equation(4) is mathematically and epidemiologically well posed in D . Let \dot{D} represent the interior of D .

Theorem 3.:The region $D \subset \mathbb{R}_+^3$ is positively-invariant for the basic model (4) with non-negative initial conditions in \mathbb{R}_+^3 .

3.3 Equilibrium solution

The system of equation (4) has only one equilibrium solution i.e the disease free equilibrium solution. $M_0 = (\frac{\gamma}{\beta}, 0, 0)$

Theorem 4.:The disease free equilibrium (DFE) of the malaria model(4) is locally asymptotically stable(LAS) if $R_0 < 1$, and unstable if $R_0 > 1$.

3.4 Further Analysis of the system

Since $S(t) + I(t) + R(t) = 1$, we can compute $R(t)$ from $R(t) = 1 - S(t) - I(t)$ by writing the second equation in (4) as

$$\frac{dI}{dt} = \beta SI - \gamma I = \beta(S - \frac{\gamma}{\beta})I$$

, Hence,

$$\frac{dI}{dt} = \beta(S - \frac{\gamma}{\beta})I$$

Proposition 1:If $S_0 < \frac{\gamma}{\beta}$, then $\frac{dI}{dt} < 0$

Proof:Since

$$\frac{dI}{dt} = \beta(S - \frac{\gamma}{\beta})I$$

If $S_0 < \frac{\gamma}{\beta}$ then $S_0 = -\frac{\gamma}{\beta}$.

$$\begin{aligned} \therefore \frac{dI}{dt} &= \beta(-\frac{\gamma}{\beta} - \frac{\gamma}{\beta})I \\ &= \beta(-\frac{2\gamma}{\beta})I \\ &= -2\gamma I < 0 \\ \therefore \end{aligned}$$

$\frac{dI}{dt} < 0$, So $I(t)$ decreases and disease dies out

Proposition 2: If $S_0 > \frac{\gamma}{\beta}$, then $\frac{dI}{dt} > 0$

Proof: Since

$$\frac{dI}{dt} = \beta(S - \frac{\gamma}{\beta})I$$

If $S_0 > \frac{\gamma}{\beta}$ then $S_0 = 1 + \frac{\gamma}{\beta}$.

\therefore

$$\frac{dI}{dt} = \beta(1 + \frac{\gamma}{\beta} - \frac{\gamma}{\beta})I$$

$$= \beta I > 0$$

$$\frac{dI}{dt} = \beta I > 0$$

\therefore

$$\frac{dI}{dt} > 0$$

, So $I(t)$ increases and this result into an epidemic.

Proposition 3: $\frac{dI}{dS} = -\frac{\beta S - \gamma I}{\beta S I} = -1 + \frac{m}{S}$ where $m = \frac{\gamma}{\beta}$

Proof: Suppose

$$\frac{dS}{dt} = -\beta S I$$

,

$$\frac{dI}{dt} = \beta(S - \frac{\gamma}{\beta})I$$

$$\therefore \frac{\frac{dI}{dt}}{\frac{dS}{dt}} = -\frac{\beta S I - \gamma I}{\beta S I} = -\frac{\beta S I}{\beta S I} + \frac{\gamma I}{\beta S I} = -1 + \frac{\gamma}{\beta S} \quad (\text{Let } m = \frac{\gamma}{\beta})$$

$$\frac{dI}{dS} = -1 + \frac{m}{S}$$

Proposition 4: Suppose $\frac{dI}{dS} = -1 + \frac{m}{S}$ then $I(t) = I_0 - S(t) + S_0 + m \log \frac{S(t)}{S_0}$.

Proof: $\frac{dI}{dS} = -1 + \frac{m}{S}$, where $m = \frac{\gamma}{\beta}$ $\int dI = \int_0^t (-1 + \frac{m}{S}) ds$

$$I(t) = \int -dS + \int_0^t \frac{m}{S} dS$$

$$I(t) = -S(t) + m \log_e S(t) + C \text{ at } t = 0$$

$$I_0 = -S_0 + m \log_e S_0 + C$$

$$\therefore C = I_0 + S_0 - m \log_e S_0.$$

Hence we obtain

$$I(t) = -S(t) + m \log_e S(t) + I_0 + S_0 - m \log_e S_0$$

$$I(t) = I_0 - S(t) + S_0 + m \log_e S(t) - m \log_e S_0$$

$$I(t) = I_0 - S(t) + S_0 + m \log_e \frac{S(t)}{S_0}$$

3.5 The Basic Reproduction Number

From the Proposition 1 we observed that $I(t)$ decreases when $S < \frac{\gamma}{\beta}$, $S(0) < \frac{\gamma}{\beta}$ implies that $\frac{\beta}{\gamma} S(0) < 1$ implies $\frac{\beta}{\gamma} < 1$. (i.e. $S_0 \simeq 1$). Also from the Proposition 2, we observed that $I(t)$ increases as $S(t) > \frac{\gamma}{\beta}$ implies that $\frac{\beta S_0}{\gamma} > 1$ implies that $\frac{\beta}{\gamma} > 1$. We define

$$R_0 = \frac{\beta}{\gamma}$$

.The basic reproduction number helps to determine when an infectious disease dies out or when it spreads.If $R_0 > 1$, then disease dies out without any intervention but the disease spreads out when $R_0 > 1$. When $R_0 = \frac{\beta}{\gamma} < 1$ implies $\beta < \gamma$, this means that the transmission rate is greater than the recovery rate an this results in epidemic.

$R_0 = \frac{\beta}{\gamma} = \beta \times \frac{1}{\gamma}$ is the product of the contact rate(transmission rate) per unit time and the average infectious period $\frac{1}{\gamma}$ which means that R_0 is the average number of minimum contacts a particular infective make with both susceptible and infected persons during his entire infectious period.

3.6 Global Stability

Theorem 4: Suppose $R_0 < 1$, then the disease free equilibrium M_0 is globally asymptotically stable on D .

Proof: Given that $R_0 < 1$, then there exist only the disease free equilibrium(DFE) $M_0 = (S^*, I^*, R^*) = (\frac{\gamma}{\beta}, 0, 0)$. Suppose the Lyapunov function $A(S, I, R) : \mathbb{R}^3 \rightarrow \mathbb{R}^+$ is defined as

$$A(S, I, R) = mI$$

, $m \geq 0$ By differentiating $A(S, I, R)$ with respect to time yields

$$\begin{aligned} \frac{dA}{dt} &= m \frac{dI}{dt} \\ m &\geq 0 \end{aligned}$$

By substituting into the model equation(4) yields

$$\begin{aligned} \frac{dA}{dt} &= m(\beta SI - \gamma I) \\ &= mI(\beta S - \gamma) \\ &= mI\gamma\left(\frac{\beta}{\gamma}S - 1\right) \\ &= mI\gamma(R_0S - 1) \\ &= m(R_0 - 1)I \leq 0 \end{aligned}$$

where

$$S \simeq 1$$

Hence $R_0 < 1$. Note that $\frac{dA}{dt} = 0$ only when $I = 0$. The maximum invariant set in $\{(S, I, R) \in D \mid \frac{dA}{dt} \leq 0\}$ is singleton set $\{M_0\}$. Therefore, the global stability of M_0 when $R_0 \leq 1$ is obtained from Lasalle's invariance principles.

4 Homotopy Analysis Approach

We hereby present below the procedure for the HAM (Homotopy Analysis Method) for the benefit of finding the numerical solution of our model. Consider a nonlinear equation of the form

$$A[v(t)] = 0 \tag{8}$$

where A is a linear operator, t denotes the time and $v(t)$ is an unknown function. Let $v_0(t)$ denote an initial approximation of $v(t)$ and Z denote an auxilliary linear operator Liao[57]construct the zero-order deformation equation.

$$(1 - q)Z[\varphi(t; q) - v_0(t)] = qh_1H(t)A(t; p) \quad (9)$$

where $q \in [0, 1]$ is the embedding parameter, $h \neq 0$ is a non-zero auxilliary function. When $q = 0$ and $q = 1$, the zero-order deformation equation becomes respectively

$$\varphi(t; 0) = v_0(t) \quad (10)$$

and

$$\varphi(t; 1) = v(t) \quad (11)$$

Thus, as q increases from 0 to 1, the solution $\varphi(t; q)$ varies continuously from the initial approximation $v_0(t)$ to the exact solution $v(t)$. Such a kind of continuous variation is called deformation in topology. Expanding $\varphi(t; p)$ by Taylor series in power series of q , we have

$$\varphi(t; q) = v_0(t) + \sum_{m=1}^{\infty} v_m q^m \quad (12)$$

where

$$v_m(t) = \frac{1}{m!} \frac{\partial^m \varphi(t; q)}{\partial q^m} \quad (13)$$

is the deformation derivative. If the auxilliary linear operator A , the initial approximation $v_0(t)$, the auxilliary parameter h_1 and the auxilliary function $H(t)$ are properly chosen so that

1. the solution $\varphi(t; q)$ of the zero-order deformation equation(8) exists for all $q \in [0, 1]$
2. the deformation derivative (12) exists for all $m = 1, 2, \dots$
3. the series (11) converge at $q = 1$

Then, we have the series solution

$$\varphi(t; 1) = v_0(t) + \sum_{m=1}^{\infty} v_m(t) \quad (14)$$

Define the vector

$$\vec{v}_m(t) = \{v_0(t), v_1(t), \dots, v_m(t)\} \quad (15)$$

According to the definition (12) the governing equation can be derived from the zero-order deformation equation(8). Differentiating(8) m -times with respect to the embedding parameter q , then by setting $q = 0$ and finally dividing by $m!$, we have the m th order deformation equation

$$Z[b_m(t) - \lambda_m v_{m-1}(t)] = hH(t)P_m(\vec{v}_{m-1}(t)) \quad (16)$$

where

$$P_m(\vec{v}_{m-1}(t)) = \frac{1}{(m-1)!} \frac{\partial^{m-1} A[\varphi(t; q)]}{\partial q^{m-1}} \quad (17)$$

$$\lambda_m = \begin{cases} 0 & \text{if } m \leq 1, \\ 1 & \text{if } m > 1, \end{cases} \quad (18)$$

Note that according to the definition (16), the right hand side of (15) depends only on $v_{m-1}(t)$. Thus, we easily gain the series $v_1(t), v_2(t), \dots$ by solving the linear higher-order deformation equation (15) using the well known symbolic computation software such as MAPLE, MATLAB or MATHEMATICA.

5 Solution of the SIR Model by HAM

To solve the model equation (6) by HAM, we consider the first equation in the model equation (6) and choose the linear operator

$$A[S(t; q)] = \frac{dS(t; q)}{dt} \quad (19)$$

with the property that

$$A[\alpha_1] = 0 \quad (20)$$

where α_1 is a constant of integration. The inverse operator A^{-1} is given by

$$A^{-1}(\cdot) = \int_0^t (\cdot) dt \quad (21)$$

Let the nonlinear operator be defined as

$$A[S(t; q)] = \frac{dS(t; q)}{dt} - \beta S(t; q)I(t; q) \quad (22)$$

By constructing the zero-order deformation equation

$$(1 - q)A[S(t; q) - S_0(t; q)] = qh_1H(t)A[S(t; q)] \quad (23)$$

we have the following:

1. If $q = 0$, then $S(t; 0) = S_0(t)$
2. If $q = 1$, then $S(t; 1) = S(t)$.

Therefore, we have the m th order deformation equation

$$A[S_{h,m}(t) - \lambda S_{(m-1)}(t)] = h_1H(t)P(\bar{S}_{(m-1)}(t)), m \geq 1 \quad (24)$$

where

$$P_m(\bar{S}_{(m-1)}(t)) = \frac{dS_{(m-1)}(t)}{dt} - \beta S(t)I(t) \quad (25)$$

The solution of the m th order deformation equation (24) for $m \geq 1$ and using $h_1 = -1$ and $H(t) = 1$ is given by

$$S_m(t) = \lambda_m S_{(m-1)}(t) - \int_{\infty}^t \left[\frac{dS_{(m-1)}(t)}{dt} + \beta \sum_{k=0}^{m-1} S_k(t)I_{m-1-k}(t) \right] dt, m \geq 1 \quad (26)$$

Following earlier steps, we obtain

$$I_m(t) = \lambda_m I_{(m-1)}(t) - \int_{\infty}^t \left[\frac{dI_{(m-1)}(t)}{dt} - \beta \sum_{k=0}^{m-1} S_k(t)I_{m-1-k}(t) + \gamma I_{m-1}(t) \right] dt, m \geq 1 \quad (27)$$

$$R_m(t) = \lambda_m R_{m-1}(t) - \int_{\infty}^t \left[\frac{dR_{m-1}(t)}{dt} - \gamma I_{m-1}(t) \right] dt, m \geq 1 \quad (28)$$

6 Numerical Results and Discussions

We execute the numerical analysis of the model using the parameters obtained from different literatures. The table below shows the details of the parameters and their values.

Parameter	Symbol	Value	Source
rate at which the infected become immunized/isolated	γ	0.02	[3]
rate of transmission	β	0.01	[3]

Table 2: Table showing numerical values of parameters used in the simulations.

Variables	Symbol	Value	Source
Initial population of susceptible	S_0	20	[3]
Initial population of Infected	I_0	15	[3]
Initial population of Recovered	R_0	10	[3]

Table 3: Table showing initial values of variables used in the simulations.

The 3rd to 9th approximations for S, I, R , are obtained and presented below:

3rd approximations:

$$\begin{aligned} S_3(t) &= 20 - 3t - 0.015t^2 + 0.02625t^3 \\ I_3(t) &= 15 + 2.4t + 0.018t^2 - 0.02655t^3 \\ R_3(t) &= 10 + 0.3t + 0.024t^2 + 0.0003t^3 \end{aligned}$$

4th approximations:

$$\begin{aligned} S_4(t) &= 20 - 3t - 0.015t^2 + 0.02355t^3 + 0.00098437t^4 \\ I_4(t) &= 15 + 2.4t + 0.018t^2 - 0.02367t^3 - 0.000851625t^4 \\ R_4(t) &= 10 + 0.3t + 0.024t^2 + 0.00012t^3 - 0.00013275t^4 \end{aligned}$$

5th approximations:

$$\begin{aligned} S_5(t) &= 20 - 3t - 0.015t^2 + 0.02355t^3 + 0.000502875t^4 - 0.00029246625t^5 \\ I_5(t) &= 15 + 2.4t + 0.018t^2 - 0.02367t^3 - 0.000384525t^4 + 0.00029587275t^5 \\ R_5(t) &= 10 + 0.3t + 0.024t^2 + 0.00012t^3 - 0.00011835t^4 - 0.0000034065t^5 \end{aligned}$$

6th approximations:

$$\begin{aligned} S_6(t) &= 20 - 3t - 0.015t^2 + 0.02355t^3 + 0.000525375t^4 - 0.00025422525t^5 - 0.00001519858125t^6 \\ I_6(t) &= 15 + 2.4t + 0.018t^2 - 0.02367t^3 - 0.000407025t^4 + 0.00025576335t^5 + 0.00001421233875t^6 \\ R_6(t) &= 10 + 0.3t + 0.024t^2 + 0.00012t^3 - 0.00011835t^4 - 0.0000015381t^5 + 0.0000009862425t^6 \end{aligned}$$

7th approximations:

$$\begin{aligned} S_7(t) &= 20 - 3t - 0.015t^2 + 0.02355t^3 + 0.000525375t^4 - 0.00025422525t^5 - 0.00000689593875t^6 + 0.000000319332367t^7 \\ I_7(t) &= 15 + 2.4t + 0.018t^2 - 0.02367t^3 - 0.000407025t^4 + 0.00025562835t^5 + 0.0000604339425t^6 - 0.000003233930352t^7 \\ R_7(t) &= 10 + 0.3t + 0.024t^2 + 0.00012t^3 - 0.00011835t^4 - 0.0000016281t^5 + 0.0000008525445t^6 + 0.00000004060668214t^7 \end{aligned}$$

8th approximations:

$$\begin{aligned} S_8(t) &= 20 - 3t - 0.015t^2 + 0.02355t^3 + 0.000525375t^4 - 0.00025400025t^5 - 0.00000761706375t^6 + 0.00000272910846t^7 + 0.000000021927188141t^8 \\ I_8(t) &= 15 + 2.4t + 0.018t^2 - 0.02367t^3 - 0.000407025t^4 + 0.00025562835t^5 + 0.0000676496925t^6 - 0.000002746375301t^7 - 0.000000211870555t^8 \\ R_8(t) &= 10 + 0.3t + 0.024t^2 + 0.00012t^3 - 0.00011835t^4 - 0.0000016281t^5 + 0.0000008520945t^6 + 0.00000001726664071t^7 - 0.00000000808482588t^8 \end{aligned}$$

9th approximations:

$$\begin{aligned} S_9(t) &= 20 - 3t - 0.015t^2 + 0.02355t^3 + 0.000525375t^4 - 0.00025400025t^5 - 0.00000760581375t^6 + 0.000002708512281t^7 + 0.0000000883349536t^8 - 0.00000003417658994t^9 \\ I_9(t) &= 15 + 2.4t + 0.018t^2 - 0.02367t^3 - 0.000407025t^4 + 0.00025562835t^5 + 0.0000675371925t^6 - 0.000002727840765t^7 - 0.0000000814690153t^8 + 0.00000000346458945t^9 \\ R_9(t) &= 10 + 0.3t + 0.024t^2 + 0.00012t^3 - 0.00011835t^4 - 0.0000016281t^5 + 0.0000008516445t^6 + 0.0000000401135786t^7 - 0.000000014950764131t^8 - 0.0000000046930456781t^9 \end{aligned}$$

7 Conclusion

In this work, we have studied the qualitative and quantitative analysis of a three-compartmental deterministic mathematical model rigorously. The Homotopy Analysis approach has been employed to approximately solve the system of nonlinear equations of SIR dynamics in particular. It was observed from our

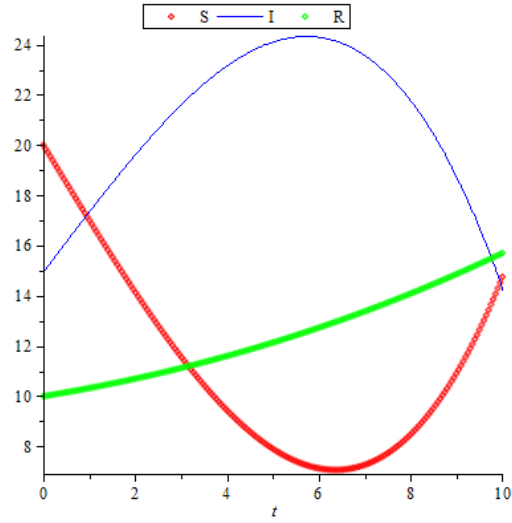


Figure 1: In the figure (1) above, we have the plot of 3^{rd} approximation for S, I, R , against time.

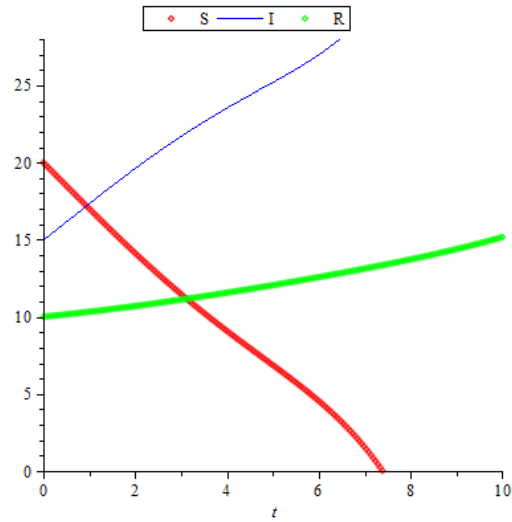


Figure 2: In the figure (2) above, we have the plot of 6^{th} approximation for S, I, R against time

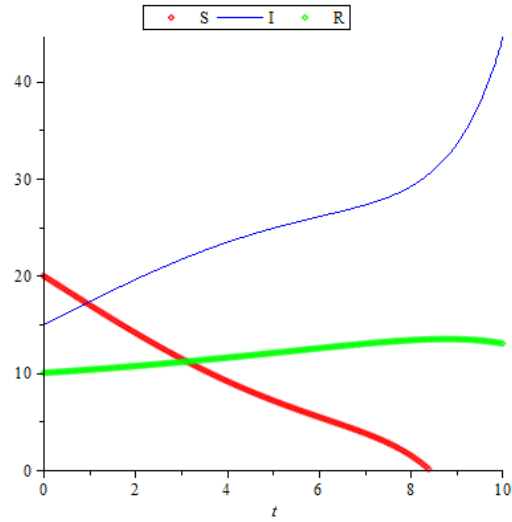


Figure 3: In the figure (3) above, we have the plot of 9th approximation for S, I, R against time

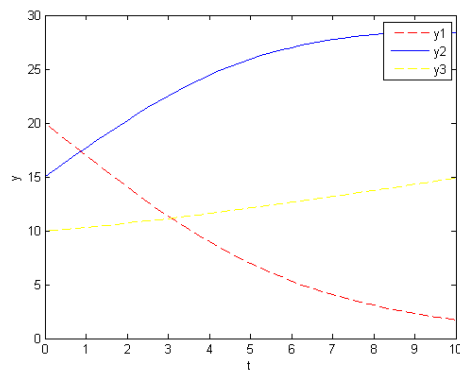


Figure 4: In the figure (4) above, is the plot obtain by using ode45 approximation for S, I, R , against time.

results that the potential and efficiency of Homotopy Analysis method in solving nonlinear problems is very powerful and reliable. With this method one is safe from the hardship and heavy computational work involved in finite-difference method and parallel techniques.

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